Magnesium promotes osteogenesis of mesenchymal stem cells via Notch signaling

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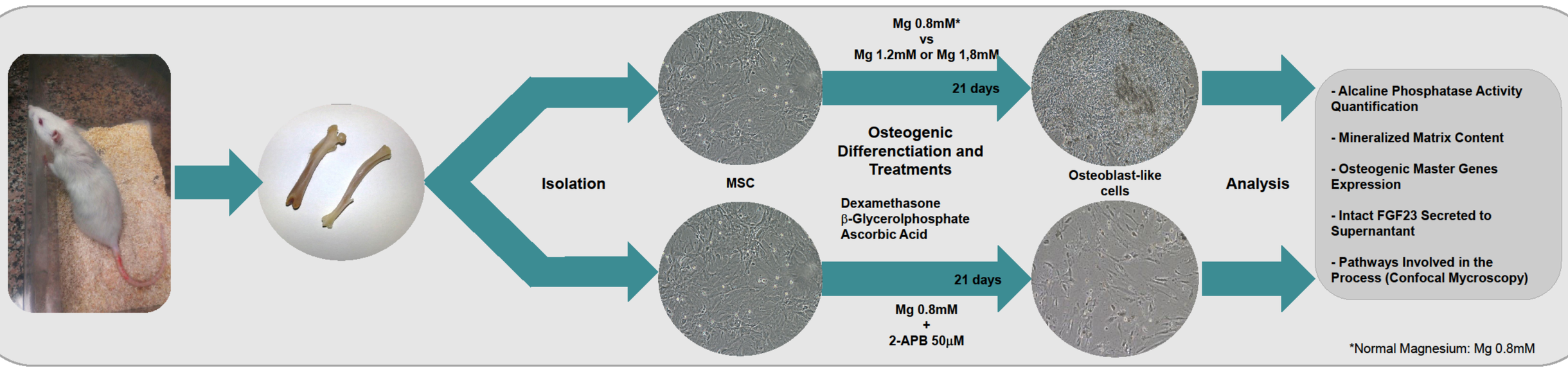
INTRODUCTION

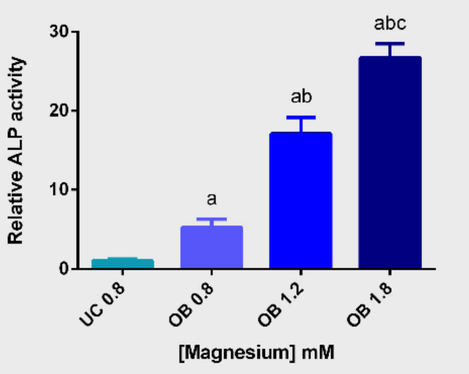
Magnesium is the fourth most abundant cation in the body and is mainly stored in bone[1,2]. Hypomagnesaemia is associated with low mineral density in the general population, whereas for patients with chronic kidney disease, controversial data has been published[3,4]. In the following study the effects of elevated magnesium concentrations on osteogenic differentiation of rat mesenchymal stem cell (MSC) were assessed to further elucidate its role in bone homeostasis. Furthermore, the inhibition of the magnesium transporter TRPM7 by 2-Aminoethoxydiphenyl borate (2-APB) addition was also evaluated. The effect of magnesium on mineralization, osteogenic marker genes and pro-osteogenic pathways was analyzed.

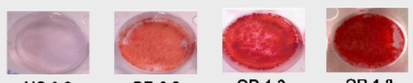
METHODS

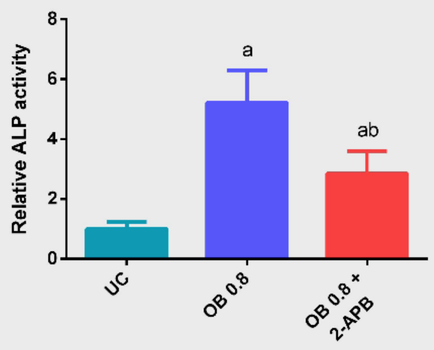


RESULTS

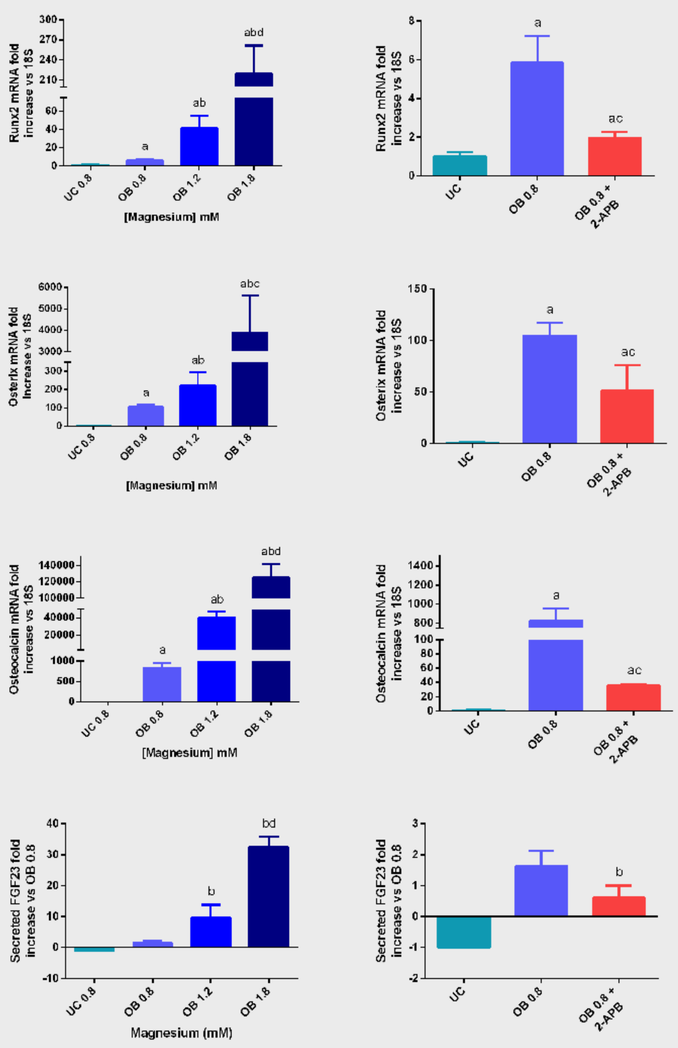
Mineralization



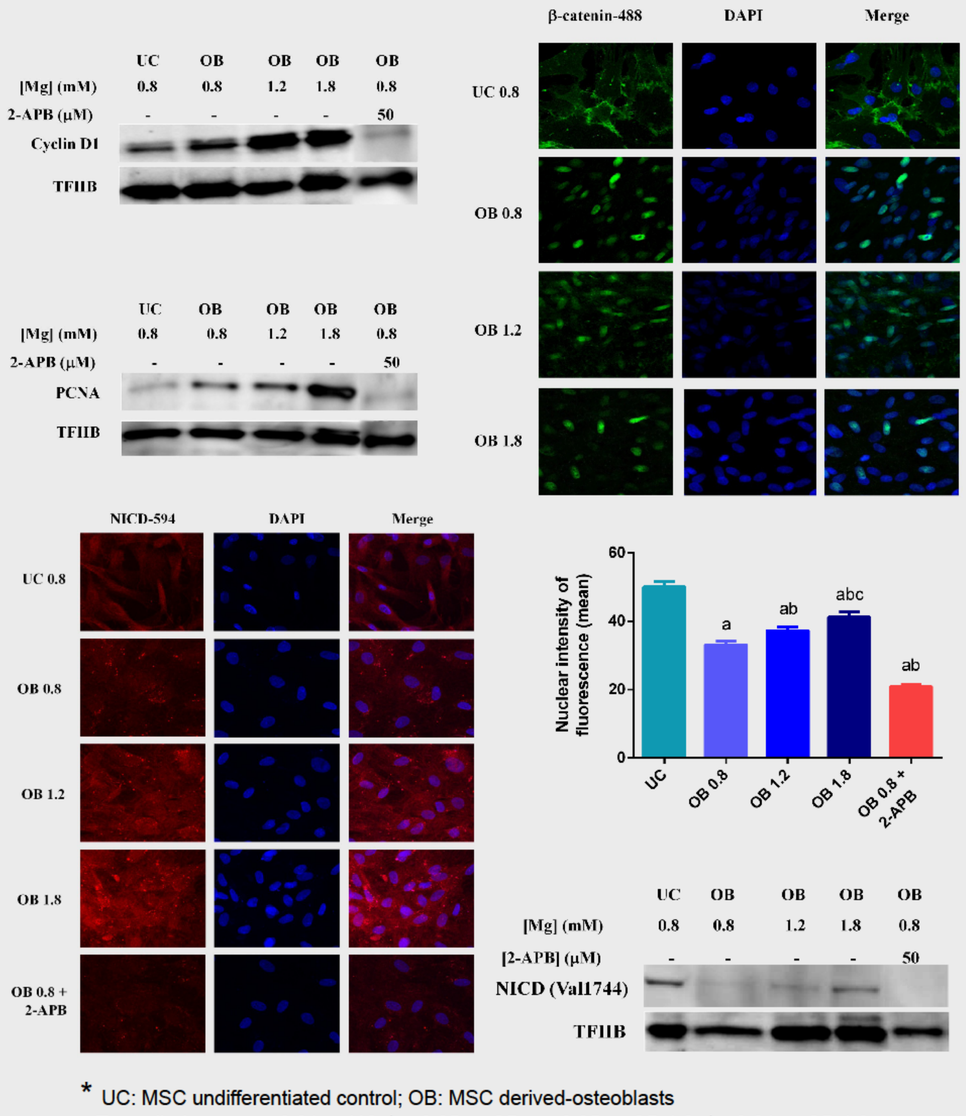




Genes Expression & FGF23 Secretion



Proliferation & Pathways



* UC: MSC undifferentiated control; OB: MSC derived-osteoblasts
Two tailed t-test: [a]p<0,01 vs UC; [b]p<0,05 vs OB; [c]p<0,05 vs OB 1.2; [d]p<0,01 vs OB 1.2

CONCLUSIONS

In conclusion our results show that magnesium directly enhances osteogenesis likewise promotes proliferation of osteoblast progenitors cells via Notch signaling. Magnesium may be beneficial for the bone metabolism and it is not directly responsible of an adynamic bone.

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201--MP
H) Clinical Nephrology, CKD. H1) Lab methods, progression & risk factors for CKD 1-5, nutrition in CKD, renal diseases (except GNs and cystic diseases).
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